CASE REPORT

JIACM 2026; 27 (3): 220-22

Severe Haemotoxic Viper Envenomation with Recurrent VICC, Transient Myocardial Injury, Dynamic Conduction Abnormalities, and Kounis Syndrome following Antivenom Therapy

Himanth Erappra, SK Singh**, Varghese Koshy****

Abstract

Viper envenomation is predominantly associated with venom-induced consumption coagulopathy (VICC), local tissue injury, and acute kidney injury, while cardiovascular complications are uncommon but potentially life-threatening. We report a 33-year-old previously healthy male who developed severe haemotoxic envenomation following a viperid snakebite, with rapidly progressive local swelling and VICC. Following administration of polyvalent anti-snake venom (ASV), he developed severe anaphylaxis requiring intramuscular adrenaline. Subsequently, he developed acute chest pain with transient ST-segment elevation in leads II, III and aVF and positive cardiac troponin, with spontaneous resolution of the ischaemic changes, raising the possibility of Kounis syndrome. Serial electrocardiograms demonstrated dynamic conduction abnormalities progressing from 2:1 atrioventricular block to complete heart block and broad-complex bradyarrhythmia, followed by adenosine-responsive supraventricular tachycardia. His course was further complicated by hypotension, hyperkalaemia, oliguria, and acute kidney injury. Recurrent VICC necessitated repeated ASV administration, with a cumulative dose of 46 vials. With supportive and multidisciplinary management, coagulation abnormalities, renal dysfunction, local swelling, and cardiac rhythm disturbances progressively resolved, although residual left anterior fascicular block persisted at discharge. This case highlights the rare coexistence of recurrent VICC, transient myocardial injury, dynamic conduction abnormalities, supraventricular tachycardia, acute kidney injury, and probable Kounis syndrome following ASV-induced anaphylaxis. Severe viper envenomation warrants continuous cardiac monitoring with serial electrocardiography and cardiac biomarker assessment, particularly when chest pain or arrhythmias develop during antivenom therapy.

Key words: Viper bite, venom-induced consumption coagulopathy, Kounis syndrome, complete heart block, myocardial injury, snake envenomation.

Introduction

Snakebite envenomation remains a major public health problem in tropical countries, particularly India, where viperid snakes contribute substantially to morbidity and mortality. Haemotoxic envenomation commonly results in venom-induced consumption coagulopathy (VICC), bleeding manifestations, acute kidney injury, and local tissue injury[1]. Cardiac involvement is increasingly recognised but remains poorly characterised. Reported cardiovascular complications include myocardial infarction, coronary vasospasm, myocarditis, conduction abnormalities, arrhythmias, and cardiogenic shock[2]. These manifestations may result from direct venom cardiotoxicity, endothelial injury, hypercoagulability, coronary thrombosis, or allergic reactions associated with antivenom therapy[3]. Kounis syndrome, defined as acute coronary syndrome precipitated by allergic or hypersensitivity reactions, is a particularly rare but important differential diagnosis[4].

We describe a case of severe viperine envenomation complicated by recurrent VICC, transient myocardial ischaemia, dynamic conduction abnormalities, and probable Kounis syndrome.

Case Presentation

A 33-year-old serving soldier with no known comorbidities presented to a military hospital in Nagaland following a snakebite sustained over the dorsum of the right index finger at approximately 0530 hours on 03 June 2026 while working within unit premises. The offending snake was killed and photographed shortly after the incident and was morphologically consistent with a viperid species. Soon after the bite, he developed rapidly progressive painful swelling involving the right index finger extending to the wrist. On admission, fang marks were visible approximately 2 cm below the nail bed. Examination revealed diffuse swelling, erythema, and local warmth involving the affected digit without evidence of neurotoxic manifestations such as ptosis, dysarthria, dysphonia, or respiratory compromise.

Initial laboratory investigations demonstrated evidence of haemotoxic

Senior Resident, **Professor and Unit Head, ***Professor, Department of Internal Medicine and Rheumatology, Armed Forces Medical College, Pune - 411 040, Maharashtra.

Corresponding Author: Dr SK Singh, Senior Resident, Professor and Unit Head, Department of Internal Medicine, Armed Forces Medical College, Pune - 411 040, Maharashtra. Tel: 8092665282, E-mail: santoshsingh4006@gmail.com

envenomation with abnormal clotting parameters suggestive of VICC. Baseline haemoglobin was 14.4 g/dL, platelet count was preserved, and serum creatinine was 1.0 mg/dL. D-dimer levels were markedly elevated at 5,139 ng/mL and subsequently increased to >10,000 ng/mL. Given severe haemotoxic envenomation and a positive 20-minute whole blood clotting test (20WBCT), treatment was initiated with a loading dose of 10 vials of polyvalent anti-snake venom (ASV) within 06 hrs of snakebite. Shortly after receiving approximately 10 mL of reconstituted ASV, the patient developed severe anaphylaxis characterised by acute bronchospasm. He was successfully managed with intramuscular adrenaline and supportive care. After stabilisation, ASV infusion was cautiously resumed at a slower rate under close monitoring.

During ASV administration, the patient developed acute central chest pain. Simultaneous ECG demonstrated ST-segment elevation in leads II, III, and aVF with reciprocal ST-segment depression in lead V1. Cardiac troponin was positive. Differential diagnoses considered included acute coronary syndrome, coronary vasospasm, venom-induced myocardial injury, and Kounis syndrome secondary to antivenom-induced anaphylaxis. The ischaemic ECG changes subsequently resolved. Despite resolution of chest pain and ST-segment abnormalities, serial ECGs revealed progressive conduction disturbances. The patient developed 2:1 atrioventricular block which progressed to complete heart block and symptomatic broad-complex bradyarrhythmia. Intravenous atropine was administered according to Advanced Cardiac Life Support protocols. Following atropine administration, he developed supraventricular tachycardia with rapid ventricular response that successfully reverted following administration of 6 mg intravenous adenosine. The clinical course was further complicated by hypotension (lowest recorded blood pressure 88/46 mmHg), worsening local envenomation extending to the elbow, hyperkalaemia, oliguria, and AKI with serum creatinine rising from 1.0 mg/dL to 1.3 mg/dL. The patient required inotropic support, anti-hyperkalaemic therapy, broad-spectrum antibiotics, repeated ASV administration, and intensive monitoring. Temporary pacing was considered but deferred because rhythm abnormalities improved with medical management. At approximately 12 hours after the bite (Day 0), the patient was clinically stabilised following supportive management.

Persistent and recurrent VICC necessitated repeated ASV dosing. The patient ultimately received a cumulative dose of approximately 46 vials of polyvalent ASV. Serum fibrinogen reached 189 mg/dL and prothrombin time remained mildly prolonged. Despite severe coagulation abnormalities, no clinically significant spontaneous bleeding occurred and platelet counts remained preserved throughout hospitalisation. Over subsequent days, renal function normalised, coagulopathy resolved, local swelling improved, and cardiac rhythm disturbances subsided. Follow-up ECG demonstrated residual left anterior fascicular block (LAFB). The patient was discharged in a haemodynamically stable condition by 06 June 26 and referred to a tertiary cardiac center for coronary angiography and further evaluation of myocardial injury.

Discussion

Viper envenomation is characterised by complex haematological and vascular effects mediated by procoagulant toxins, metalloproteinases, phospholipases, and endothelial toxins[5]. VICC remains the hallmark of severe haemotoxic envenomation and may recur despite initial correction owing to ongoing venom redistribution. The present case is unique for the coexistence of several uncommon cardiovascular complications:-

i. Transient Myocardial Injury: The patient developed acute chest pain, positive cardiac biomarkers, and transient inferior ST-segment elevation. potential mechanisms include:-
   a. Direct venom cardiotoxicity
   b. Coronary vasospasm
   c. Microvascular thrombosis

Table I: Serial haematological, biochemical, coagulation, and electrocardiographic findings following viper envenomation.

Investigation/ECG	03 Jun 2026 (Day 0)	04 Jun 2026 (Day 1)	05 Jun 2026 (Day 2)	08 Jun 2026 (Day 5)
Hb (g/dL)	14.4	13.3	–	14.3
TLC (/mm^3)	6,600	12,700	>16,500	9,800
Platelets (lakh/mm^3)	3.73	2.94	>3.11	3.53
Urea/Creatinine (mg/dL)	34/1.0	37/1.3	–	32/1.1
Na/K (mEq/L)	135/4.3	145/5.3	–	138/4.1
D-dimer (ng/mL)	5,139	8,864	>10,000	431
Fibrinogen (mg/dL)	–	189	–	–
INR	1.3	1.2	–	–
ECG	Baseline	ST elevation II, III, aVF – 2:1 AV block	CHB – broad-complex bradyarrhythmia – SVT	LAFB

d. Demand ischaemia

e. Kounis syndrome

The spontaneous resolution of ECG changes without reperfusion therapy favours coronary vasospasm or allergic coronary syndrome over thrombotic myocardial infarction.

ii. Kounis Syndrome: Kounis syndrome is defined as acute coronary syndrome occurring in association with mast-cell activation and allergic reactions[6]. The temporal relationship between ASV-induced anaphylaxis and subsequent ischaemic ECG changes strongly supports this possibility. To our knowledge, reports of Kounis syndrome following antivenom administration remain exceedingly rare.

iii. Conduction System abnormalities: The evolution from 2:1 AV block to complete heart block and broad-complex bradyarrhythmia is particularly unusual. Proposed mechanisms include:-

a. Direct venom-mediated myocardial toxicity

b. Ischaemia of the conduction system

c. Myocarditis-like inflammatory injury

d. Electrolyte disturbances

e. Autonomic dysfunction

The subsequent recovery without permanent pacing suggests reversible toxin-mediated injury.

iv. Supraventricular Tachycardia: Development of SVT following atropine administration may reflect heightened sympathetic activity in a myocardium already sensitised by venom-induced injury.

v. Recurrent VICC: Persistent elevation of D-dimer levels (>10,000 ng/mL) and recurrent clotting abnormalities despite initial treatment illustrate delayed or recurrent VICC, a phenomenon increasingly recognised in severe viper envenomation.

An important learning point from this case is the simultaneous occurrence of multiple reversible cardiovascular complications in the setting of severe hemotoxic envenomation. While myocardial injury, atrioventricular conduction disturbances, supraventricular tachyarrhythmias, recurrent venom-induced consumption coagulopathy (VICC), and Kounis syndrome have each been reported individually, their coexistence in a single patient is exceedingly rare. The temporal sequence of events suggests that several overlapping mechanisms – including direct venom cardiotoxicity, endothelial dysfunction, transient coronary vasospasm, systemic inflammatory response, electrolyte abnormalities, and mast cell-mediated hypersensitivity following antivenom administration – may have acted synergistically. This case also highlights the diagnostic challenge of distinguishing acute coronary thrombosis from venom-related myocardial injury or allergic coronary syndrome, as management strategies differ substantially. The complete recovery of cardiac rhythm abnormalities without permanent pacing further supports the reversible nature of toxin-mediated conduction system injury when recognized early and managed appropriately. Therefore, patients with severe haemotoxic snakebite should undergo serial electrocardiography, cardiac biomarker assessment, and continuous cardiac monitoring, particularly following antivenom administration or the development of chest pain, as prompt recognition and supportive treatment may prevent potentially fatal complications.

Conclusion

Severe viper envenomation may present with a broad spectrum of cardiovascular manifestations extending beyond coagulopathy and renal injury. This case demonstrates the rare coexistence of recurrent VICC, probable kounis syndrome, transient myocardial injury, complete heart block, broad-complex bradyarrhythmia, supraventricular tachycardia and acute kidney injury.

Clinicians should maintain a high index of suspicion for cardiac complications in severe envenomation, particularly when chest pain, ECG abnormalities, or arrhythmias develop during treatment. Early multidisciplinary management and continuous cardiac monitoring are essential for favourable outcomes.

References

1. Ojha A, Hadimani P, Anthony D *et al*. Optimizing Anti-Snake Venom Strategies for Haemotoxic Envenomation in Northern India: Clinical Outcomes and Regional Challenge. *Cureus* 17 (5): e84090.
2. A Study of Clinicobiochemical and Cardiac Profile in Snakebite at a Tertiary Care Center in Northern India [Internet]. [cited 2026 Jul 30]. Available from: https://japi.org
3. Averin AS, Utkin YN. Cardiovascular Effects of Snake Toxins: Cardiotoxicity and Cardioprotection. *Acta Naturae* 2021; 13 (3): 4.
4. Rodríguez-Ruiz C, Puig-Carrión G, Delgado-Nieves A. Kounis Syndrome: A More Commonly Encountered Cause of Acute Coronary Syndrome. *Heart Views Off J Gulf Heart Assoc* 2019; 20 (3): 122-5.
5. Khelfi A. Pathophysiological and therapeutic insights into Viperidae envenomation: Mechanisms, clinical manifestations, and advances in antivenom strategies. *Toxicol Anal Clin* 2026; 38 (2): 180-97.
6. Kounis NG. Kounis syndrome (allergic angina and allergic myocardial infarction): A natural paradigm? *Int J Cardiol* 2006; 110 (1): 7-14.